

WHITE COAT HYPERTENSION/EFFECT IS ASSOCIATED WITH INCREASED SMALL VESSEL DISEASE IN THE BRAIN

Drazich, E.¹, Vundavalli, S.¹, Kirkham, F.¹, Rankin, P.¹, Timeyin, J.¹, Bunting, E.¹, Ali, K.^{1,2}, Rajkumar, C.^{1,2}
AFFILIATIONS: ¹Department of Elderly Care and Stroke Medicine, Brighton and Sussex University Hospitals Trust, Brighton, UK

²Department of Medicine, Brighton and Sussex Medical School, Brighton, UK



OBJECTIVE

Small vessel disease, as measured by white matter hyperintensity (WMH) in the brain, is known to be associated with increased stroke risk and cognitive impairment [1,2]. Vascular stiffness is also considered an independent risk factor for stroke[3,4]. This study explored the relationship between WMH on computerised tomography (CT), vascular stiffness, and white coat hypertension/effect (WCH/E) in patients with recent transient ischaemic attack (TIA) or lacunar stroke (LS).

DESIGN AND METHOD

Ninety-six patients recruited for the ASIST trial (Arterial Stiffness in Lacunar Stroke and TIA) underwent measurement of clinic blood pressure (BP) and ambulatory BP monitoring (APBM) within two weeks of TIA or LS. Twenty-three patients had normotension (clinic BP <140/90mmHg and day-time ABPM <135/85mmHg) and 25 patients had WCH/E (clinic BP ≥140/90mmHg and day-time ABPM <135/85mmHg). Arterial stiffness was measured using carotid-femoral pulse wave velocity (PWV) (Complior®, ALAM Medical) and carotid-ankle vascular index (CAVI) (VaSera VS-1500N®, Fukuda Denshi). CT images were rated for WMH using the four-point Fazekas visual rating scale [5,6], where 0=no WMH, 1=mild WMH (small, separate white matter lesions), 2=moderate WMH (confluent joined up lesions around the ventricles), and 3=severe WMH (white matter lesions extending from ventricle into the cortex). The rater was trained on 30 images by a consultant neuroradiologist. Patients were grouped into no-mild WMH (scores 0-1) or moderate-severe (scores 2-3) groups. The relationship between BP, vascular stiffness and WMH was explored with t-tests, chi-square and logistic regression accounting for known cardiovascular risk factors.

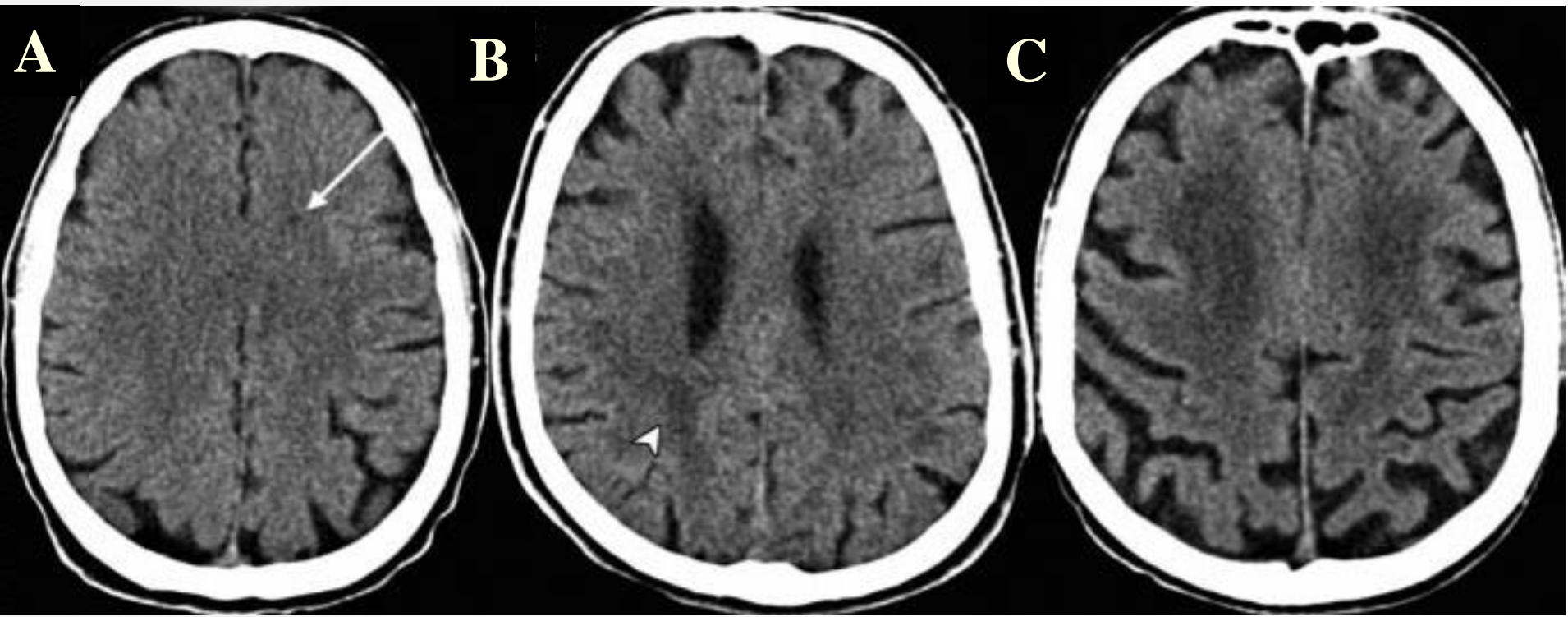


Figure 1. CT images of Fazekas 1-3. A – Fazekas 1, B – Fazekas 2, C – Fazekas 3 [7].

RESULTS

Forty-four percent of patients with WCH/E had moderate-severe WMH compared to 17% of normotensives (p=0.047). Arterial stiffness was higher in the WCH/E group compared to normotensives (CAVI 10.3 + 1.1 vs 9.2 + 1.5 p = 0.007; PWV 12.2 + 3.2 vs 9.5 + 2.3 p= 0.003). The regression model with WMH as the dependent factor, and WCH/E and cardiovascular risk factors as independent factors showed WCH/E and either CAVI or PWV to be the only independent significant factor contributing to WMH (CAVI:p=0.038, PWV:p=0.043).

Table 1: Results	Normotension (N=23)	WCH/E (N=25)	Statistical Sig.
Female n(%)	8 (35%)	7 (28%)	p=0.613
Age (years)	69.7+/-11.8	76.5+/-8.7	p=0.026
Ever Smoker n(%)	12 (52%)	18 (72%)	p=0.156
Anti-hypertensive use n(%)	15 (65%)	20 (80%)	p=0.250
Hyperlipidaemia n(%)	18 (78%)	15 (60%)	p=0.458
Diabetes n(%)	4 (17%)	7 (28%)	p=0.173
CAVI	9.2+/-1.5	10.3+/-1.1	p=0.007
PWV	9.5+/-2.3	12.2+/-3.1	p=0.003
No-Mild WMH n(%)	19 (83%)	14 (66%)	p=0.047
Moderate-Severe WMH n(%)	4 (17%)	11(44%)	

CONCLUSIONS

Patients with WCH/E were more likely to have moderate-severe WMH on CT than normotensives. WCH/E group had increased WMH, likely modulated by vascular stiffness, independent of other cardiovascular risk factors. This study suggests that WCH/E is associated with increased WMH in the brain and may benefit from treatment.

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